Combining Synthetic Controls and VARs:

On the Estimation of Causal Effects in Time Series Data

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Abstract

We argue that applications of Synthetic Control (SC) are faced with a self-selection problem. That is, the method is primarily applied to non-complex data structures that are straightforward to forecast, given the availability of donors in the post-treatment period. Using simulation studies, we show that the high interpretability of SC comes at the cost of poor predictions and forecasts, which are especially pronounced if the data generating process contains a time series structure. To address this issue, we introduce the intricacy-statistics that informs the applied researcher whether or not the data at hand exceeds a level of time series structure that SC can handle. If the case, more flexible methodologies that combine the strengths of SC and conventional time series techniques promise more accurate predictions and forecasts. Hence we introduce the new Vector Autoregressive Synthetic Control (VARSC) estimator, that takes in account both the time series structure and the availability of donors. In order to implement these ideas, we introduce the R-package varsc that provides ready-to-use functions to compute the intricacy-statistics and, based on the magnitude of the statistics, the functionalities to estimate either the SC or the VARSC model. To probe the performance of our methodology outside the experimental setting, we apply it to three existing applications of SC: Specifically, we show that our proposed model performs equally well like the SC-method. The result is striking because in contrast to the SC-model, our models gets along without the informational contend of potential covariates.

Keywords: *Synthetic Control; Causality; VAR*

List of Acronyms

ADH Abadie, Diamond, and Hainmueller

GDP Gross Domestic Product

DGP Data Generating Process

iid independent and identically distributed

MSFE Mean Squared Forecast Error

MSPE Mean Squared Prediction Error

OLS Ordinary Least Squares

PC Principal Components

SC Synthetic Control

USA United States of America

VAR Vector Autoregression

VARSC Vector Autoregressive Synthetic Control

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1. Introduction

This is work in progress

SC method is combined with Vector Autoregression (VAR). Method was introduced by Abadie, Diamond, and Hainmueller (ADH). Paper strcuture could be similar to the one by [Doudchenko and Imbens, 2016]

2. Literature Review 2-3 pages

Literature has been clustered. This is work in progress

2.1. Synthetic Control

The SC method was developed by Alberto Abadie and colleagues in a series of influential papers ([Abadie and Gardeazabal, 2003], [Abadie et al., 2010], [Abadie et al., 2015]). The method is designed to estimate the causal effect of a treatment in a setting with a single treatment unit and a number of potential control units. Pre- and post-treatment data are observed for the treatment and control units for the outcome of interest as well as for a set of covariates. The SC-procedure combines aspects of the matching and difference-in-difference literature and can therefore be interpreted as a relative of the causal inference literature introduced by [Rubin, 1974]. Similar to many other microeconometric methods, the objective is to distinguish causation from correlation and to assess the magnitude and significance of treatments in observational case studies.
In their canonical 2003 article, Abadie and Gardeazabal evaluate the causal economic effects of conflict using terrorist conflicts in the Basque Country as a comparative case study. In their specific application example, they find that terrorist conflicts caused the per capita Gross Domestic Product (GDP) of the treatment unit (Basque Country) to decline by about 10% relative to the synthesized control unit.

some more words on other findis and things they did The next appropriate setting for an application of the SC method was the introduction of a large-scale tobacco control program implemented in the state of California in the United States of America (USA) in 1988.

2.2. Overview

[Abadie, 2021] read.
[Athey and Imbens, 2016] read.

2.3. Application

[Born et al., 2019] read.

[Cho, 2020] read.

[Cunningham, 2021] read.

[Funke et al., 2020] read.

2.4. Methodological Background

[Hainmueller et al., 2011] read.

[Abadie and Imbens, 2006] not read.

[Abadie and Imbens, 2002] not read.

[Doudchenko and Imbens, 2016] read.

[Ferman, 2021] read.

[Frangakis and Rubin, 2002] not read.

[Rosenbaum and Rubin, 1983] not read

[Rubin, 1974] not read.

2.5. Extensions/ Developments

[Abadie and L'Hour, 2021] read.

[Amjad et al., 2018] read.

[Ben-Michael et al., 2021] read.

[Ben-Michael et al., 2021] not read.

[Kellogg et al., 2021] not read.

[Kuosmanen et al., 2021] not read.

[Muhlbach and Nielsen, 2019] read.

Developments

[Arkhangelsky et al., 2021] not read

[Athey et al., 2017] not read.

[Brodersen et al., 2015] read.

[von Brzeski et al., 2015] read.

[Hartford et al., 2017] read.

2.6. Testing

[Andrews, 2003] not read.

[Cattaneo et al., 2021] not read.

[Chernozhukov et al., 2019] not read.

[Chernozhukov et al., 2021] not read.

[Firpo and Possebom, 2018] not read.

[Hahn and Shi, 2017] read.

2.7. Time Series Econometrics

[Martin et al., 2012] read.

[Harvey and Thiele, 2020] read.

[Breitung and Knüppel, 2021] partially read.

3. Theory

In this chapter, we propose alternative SC-estimators to assess the magnitude of treatment effects in observational settings. To establish a general basis, we first describe the contextual environment of the estimation. Similar to the setting as introduced by ADH, we consider a framework with $J+1$ panel units indexed by $j = 0, 1, ..., J$ that are observed over a time horizon of T periods. Without loss of generality, assume that unit $j = 0$ is exposed to the treatment at period $t = T_0$ with $1 < T_0 < T$ and that there are no treatment anticipation and contamination (i.e., no spillovers in time and space). The former would be the case if the treatment affects unit $j = 0$ before T_0, the latter describes the case where some of the supposedly untreated units $j = 1, ..., J$ are contaminated as they are affected by the treatment. To contextualize these assumptions, [Abadie et al., 2010] argue that in the presence of anticipation effects, T_0 could be shifted back in time until the no anticipation assumption seem plausible. If panel units in the donor pool[1] are affected by the treatment (contamination) as it is likely in the Brexit-application, those units could be removed from the sample prior to the estimation. Our goal is to evaluate the causal effect of the treatment, the specific functional form of which remains unspecified though. This is possible because the main goal of the SC-estimation lies in the precise estimation of the counterfactual. Since the treatment scenario is empirically observable, it is not necessary to specify the specific functional form of the it (e.g. level or slope shift, fading or persistent shock).

The following chapter is structured as follows: We first describe the canonical estimation procedure as proposed by ADH. Furthermore, ADH propose a model-invariant hypothesis testing approach. As this approach is employed in the further analysis, we also give a brief overview of these principles. Next we build intuition by considering a simple static scenario with only two donor units and one treatment unit. This setting is subsequently generalized to the case with more potential donors. The main difference between our extensions and the setting of ADH is that we remove the weight constraints and that we analyze a situation without covariates. The former distinction guides us to the field of regularization to prevent our method from overfitting. The latter drastically

[1] To ensure direct comparability with the SC literature, we adopt most of the commonly used terms. For example, control group units are labeled as 'donors'.

reduces the data requirements and causes our algorithm to estimate the counterfactual with a significantly smaller information set. This fact leads us to the necessity of utilizing all available information and establishes our main contribution: The integration of multivariate time series approaches into the SC-algorithm. The theoretical derivation of this estimator completes the chapter.

3.1. ADH Case

We start by presenting the SC-method and the testing procedure as introduced by ADH. For the sake of comparability and due to its notational clarity, we borrow the employed notation of Abadie and colleagues. In terms of structural design, we build on the thorough presentation of the SC-method and the related hypothesis testing procedure by [Firpo and Possebom, 2018].

Setup

The estimation task can be constituted by the potential outcome framework as introduced by [Neyman, 1923] and elaborated by [Rubin, 1974]. Let $y^I_{j,t}$ be the (potential) outcome for unit j at point t in the presence of the intervention. Likewise, let $y^N_{j,t}$ be the (potential) outcome for j at point t in the absence of the intervention. ADH define the treatment effect of the intervention as

$$\delta_{j,t} = y^I_{j,t} - y^N_{j,t}$$

and introduce the indicator variable $D_{j,t}$ that takes on the value 1 if unit j is treated at period t and the value 0 otherwise. Given the assumed absence of anticipation and contamination, the following outcome is observed

$$y_{j,t} + D_{j,t}\delta_{j,t} = \begin{cases} y^N_{j,t} & (\text{if } j = 0 \text{ and } t < T_0) \text{ or } j \geq 1, \\ y^N_{j,t} + \delta_{j,t} & \text{if } j = 0 \text{ and } t \geq T_0 \end{cases}$$

The goal to estimate the causal treatment effect $(\delta_{0,T_0}, ..., \delta_{0,T})$ therefore boils down to the estimation of the counterfactuals of unit $j = 0$ in the post-treatment phase $(y_{0,T_0}, ..., y_{0,T})$, i.e. on what trajectory would unit $j = 0$ have been, was there no intervention. The basic idea of ADH is to estimate these counterfactuals as a weighted average of the donor outcomes using a data-driven approach to compute the weights. Intuitively, the weights are computed such that they optimally predict the outcomes and a set of time-invariant explanatory variables for the treatment unit in the pre-intervention phase, conditional on having a percentage interpretation. Thus, for the computation of the weights, we focus exclusively on the pre-intervention time periods $t \in \{1, 2, ..., T_0 - 1\}$. Subsequently, the counterfactuals are extrapolated by applying the calculated weights to the post-intervention time periods $t \in \{T_0, T_0 + 1, ..., T_1\}$.

Let $Y_j = (y_{j,1}, ..., y_{j,T_0-1})'$ be the vector of observed pre-intervention outcomes for unit j.[2] To distinguish treatment unit and donors, ADH collect the treatment unit in the $((T_0 - 1) \times 1)$-vector Y_0 and row-bind all donor unit vectors into the $((T_0 - 1) \times J)$-matrix Y_1. Moreover, a set of K time-invariant covariates of Y_j is observed for all panel units.[3] Therefore, let X_0 denote the $(K \times 1)$-vector of covariates for Y_0 and let X_1 denote the $(K \times J)$-matrix of explanatory variables for Y_1. To estimate the causal effect of the treatment, the SC-estimator estimates the counterfactuals $(\widehat{y}_{0,1}, ..., \widehat{y}_{0,T_0}, ..., \widehat{y}_{0,T})$ of the single treated unit for the pre- and post-intervation phase as

$$\widehat{y}_{0,t} = \sum_{j=1}^{J} \widehat{w}_j y_{j,t}^N \; \forall \; t \in \{1, ..., T\}$$

The weights $(\widehat{w}_1, ..., \widehat{w}_J)$ are constraint such that $\widehat{w_j} \geq 0 \; \forall \; j$ and $\sum_{j=1}^{J} \widehat{w_j} = 1$. It is worth noting that this constraint requires the counterfactuals to belong to the convex hull of the donors as otherwise, $\widehat{Y}_0$ will never match its true counterpart. [Abadie et al., 2010] argue that "the magnitude of discrepancy" should be calculated in advance of each SC-application. If the researcher finds that the pre-intervention values of $\widehat{Y}_0$ fall outside the convex hull of the donors, the usage of SC is not recommended. Formally, $(\widehat{w}_1, ..., \widehat{w}_J)$ is the solution of the following nested optimization problem:

$$\widehat{w}(v) = \arg\min_{w} \sum_{k=1}^{K} v_k \left(x_{0,k} - \sum_{j=1}^{J} w_j x_{j,k} \right)^2$$

[2] For instance, in the canonical example of [Abadie and Gardeazabal, 2003], Y_0 would be the vector of GDPs for Great Britain until the Brexit referendum.

[3] In the already mentioned Brexit-example, natural predictors of GDP are consumption, investment, government spending and net exports.

with v being an arbitrary positive definite vector of dimension $(K \times 1)$ which solve the second optimization problem:

$$\widehat{v} = \arg\min_{v} \sum_{t=1}^{T_0-1} \left(y_{0,t} - \sum_{j=1}^{J} \widehat{w}_j(v) y_{j,k} \right)^2$$

Subsequently, the causal effect of the intervention $\delta_{j,t}$ can be quantified at each time point after the intervention $t \in \{T_0, T_0 + 1, ..., T_1\}$ as the gap between observed $(y_{0,t}^N + \delta_{j,t})$ and predicted outcome $(\widehat{y}_{0,t}^N)$.

This two-step estimation procedure serves two crucial purposes: $\widehat{v}$ measures the relative importance of the K variables in X_1 to explain X_0. In contrast, the weighting vector $\widehat{w}(v)$ quantifies the relative importance of each unit in the donor pool. Summarizing the key concept of ADH, the SC-method ensures that the synthesized treatment unit is as similar as possible to the actual treatment unit with respect to the quantity of interest and a set of potential explanatory variables in the pre-treatment period. Especially in the canonical examples of SC, the quantity of interest (e.g. GDP) and the explanatory variables (e.g. consumption, investment, government spending and net exports) are interconnected by construction. Thus, observing that the SC-estimator was capable of approximating both targets significantly enhanced the methods credibility. If explanatory variables are omitted, the SC-algorithm reduces to an Ordinary Least Squares (OLS) estimation, constraint to have no constant and weakly positive coefficients that sum up to one.

Hypothesis Testing

The question of treatment effect significance arises naturally subsequent to the construction of the synthetic control. ADH propose a model-invariant non-parametric inference procedure that is based on the Exact Hypothesis Test proposed by [Fisher, 1935]. The basic idea behind such permutation tests is to compare the observed data with a number of randomly permuted versions of it, and to use the distribution of the test statistic calculated of the permuted samples to estimate the probability that the observed result occurred by chance alone.

In the context of SC, ADH consider permutations in region (i.e. panel unit) and time. Region permutations estimate the treatment effect vector $(\delta_{j,T_0}, ..., \delta_{j,T})$ for each panel unit $j \in \{0, ..., J\}$.[4] This procedure provides the researcher with the empirical $(J + 1)$-observational distribution of the treatment. Subsequently, it is possible to compare the estimated treatment vector $(\delta_{0,T_0}, ..., \delta_{0,T})$ of the truly treated unit with the J placebo-treatment vectors of the units of the donor pool. Given the estimated treatment effect for $j = 0$ is large, the null hypothesis of no treatment effect can be rejected at the significance level of one minus the percentile of $(\delta_{0,T_0}, ..., \delta_{0,T})$ in the empirical distribution.[5] Time permutations on the other hand consider only panel unit $j = 0$, permute T_0 to dates prior to the true treatment date and compute again the empirical treatment distribution. Given that $T_0 >> J$, this approach can increase the sensitivity of the test, since the theoretically feasible significance threshold of region permutation tests is determined by $\frac{1}{J}$. For both, region and time permutations, ADH condense the vector of estimated treatment effects into a precision metric like the Mean Squared Forecast Error (MSFE)[6] of the following form:

$$MSFE_j = \frac{\sum_{t=T_0}^{T} \left(\widehat{y}_{j,t}^N - y_{j,t}^N\right)^2}{T - T_0}$$

A possible problem that can occur when assessing the relative rarity of the estimated treatment effect using the procedure described above is the existence of outliers in the donor pool. In the context of region permutations, suppose that a donor region is very different from the rest such that it falls outside the convex hull of the remaining donors. Note, that this circumstance does not cause problems for the truly treated region and its synthesized counterfactual as we expect the SC-algorithm to assign a near zero weight to such an outlier. However, since the outlier itself cannot be synthesized precisely by the donor pool, both MSPE and MSFE are expected to be large. As this special feature causes the permutation test to be unreasonably conservative, ADH propose to exclude regions who are hard to predict, i.e. who have a MSPE that exceeds the MSPE of the truly treated unit to a great extent. Figure 1 visualizes the exclusion procedure in the tobacco control application of ADH.

[4] Note that it is necessary to exclude the truly treated unit from donor pool to ensure the validity of the no contamination assumption.

[5] For instance, let $J = 99$ such that treatment effects for 100 panel units can be computed. As long as the estimated treatment effect of the truly treated units belongs to the 95 largest effects (95th percentile or higher), the permutation test rejects the null hypothesis of no treatment effect at least at 5 percent.

[6] Note that ADH speak of the Mean Squared Prediction Error for dates before and after T_0. Since we consider the time span until T_0 as prediction window and the time span after T_0 as forecast window, we employ the label Mean Squared Prediction Error (MSPE) before T_0 and the label MSFE from T_0 onward.

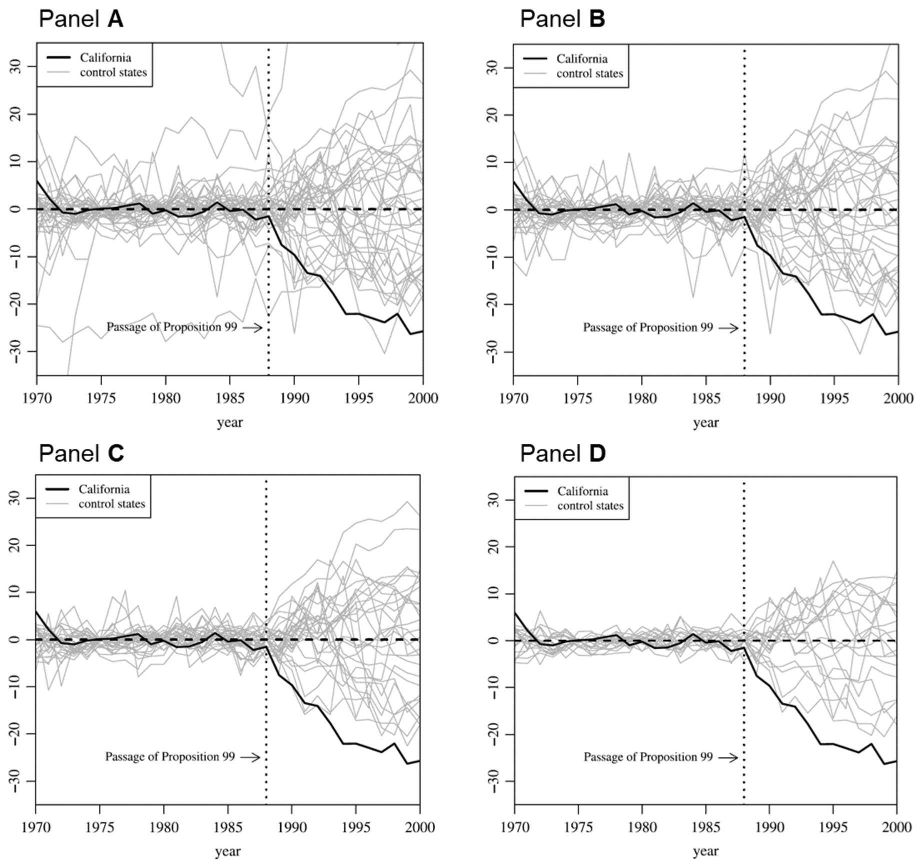

Figure 1. Region Exclusion Procedure of ADH

The vertical axis indicates the gap between observed and estimated per capita cigarette sales, the bold line represents the truly treated region (California). Two observations stand out when considering panel A: First of all, the treatment has a clear negative effect for California. Second, some regions have both a poor pre- and post-treatment fit. Since the treatment significance should not be artificially driven by regions with poor fit, ADH successively remove regions with a large MSPE relative to California. Panel B excludes regions with a MSPE that is more than 20 times as large the MSPE of California, Panel C lowers the cutoff to five times California's MSPE and Panel D to two times the MSPE. In the last scenario, only 19 regions are left and California is the one with the most extreme treatment effect. The authors therefore conclude that the treatment is statistically significant with a (permutation) p-value of 5,3% $\left(\frac{1}{19}\right)$.

One way to bypass the inefficient sample reduction procedure is to look at the distribution of the ratios of MSFE and MSPE. By scaling the post-treatment fit by the pre-treatment fit, regions with a poor fit are implicitly controlled for. In the tobacco control application, California is the region with the highest MSFE-to-MSPE ratio among all 39 regions which translates into a p-value of 2,6% $\left(\frac{1}{39}\right)$.

3.2. Simple Static Extension

Once the fundamental framework of ADH is established, we are prepared to introduce our proposed estimates. To give an intuitive introduction to our proposed extensions, we first consider the most simple scenario of one treatment unit $j = 0$ and two donor units $j = 1, 2$. We consider a setting where only the outcome series (e.g. GDP) and no further covariates (e.g. consumption, investment etc.) are observed. It is assumed that before $t = T_0$ the units have a joint distribution of the form

$$Y = \begin{pmatrix} y_0 \\ y_1 \\ y_2 \end{pmatrix} \sim \mathcal{N}(\mu, \Sigma) \text{ for } t < T_0.$$

with $\mu = (\mu_0, \mu_1, \mu_2)'$ and the positive definite covariance matrix

$$\mathbf{\Sigma} = \begin{pmatrix} \sigma_0^2 & \boldsymbol{\sigma}'_{\mathbf{12}} \\ \boldsymbol{\sigma}_{\mathbf{12}} & \mathbf{\Sigma_2} \end{pmatrix}.$$

σ_0^2 denotes the variance of y_0, $\mathbf{\Sigma_2}$ is a (2×2) covariance matrix of the vector $(y_1, y_2)'$ and $\boldsymbol{\sigma}_{\mathbf{12}}$ is a (2×1) vector with elements $cov(y_0, y_1)$ and $cov(y_0, y_2)$.

Disregarding any constraints, we are interested to derive the best unbiased forecast of y_0 given the controls y_1 and y_2 which is obtained as

$$\begin{aligned} \widehat{y}_0^N &= \mu_0 + w_1^{OLS}(y_1 - \mu_1) + w_2^{OLS}(y_2 - \mu_2) \\ &= \mu^* + w_1^{OLS} y_1 + w_2^{OLS} y_2 \end{aligned}$$

where $\mu^* = \mu_0 - w_1^{OLS}\mu_1 - w_2^{OLS}\mu_2$. This forecast can be directly estimated by an unrestricted OLS regression of y_0 on y_1 and y_1. However, the result implies that there is no inherent reason to impose the restrictions that $w_1^{OLS}, w_2^{OLS} \geq 0$ and $w_1^{OLS} + w_2^{OLS} = 1$. Furthermore, we argue that the construction of SC should include a constant term, as otherwise the estimated counterfactual may have a mean outside the convex hull of the donor means. See also [Doudchenko and Imbens, 2016] for a careful discussion of these restrictions.

For illustrative reasons, assume that

$$Y \sim \mathcal{N}\left(\begin{pmatrix} 1 \\ 1 \\ 1 \end{pmatrix}, \begin{pmatrix} 1 & 0.1 & 0.4 \\ 0.1 & 1 & 0.5 \\ 0.4 & 0.5 & 1 \end{pmatrix}\right).$$

For this example the unrestricted optimal weights for the counterfactual result as $w_1^{OLS} = -0.1333$, $w_2^{OLS} = 0.4667$ and $\mu^* = \mu_0 - w_1^{OLS} \cdot \mu_1 - w_2^{OLS} \cdot \mu_2 = 0.6667$.[7] Note that w_1^{OLS} is negative even though all bivariate correlations between the units are positive. One may argue that this result does not make much sense as the economic interpretation of y_1 entering the counterfactual $\widehat{y}_0^N$ with a negative sign is unclear. This demonstrates the trade-off between optimality in a statistical sense and the economic interpretation of the solution.

What happens if we impose the restrictions that all weights are positive and sum up to unity? In this case the restricted optimum yields the linear combination $\widetilde{y}_0^N = 0.2y_1 + 0.8y_2$. The important difference lies in the variance of these estimates. For our example we obtain

$$var(y_0 - \widehat{y}_0^N) = 0.8267$$
$$var(y_0 - \widetilde{y}_0^N) = 1.1600.$$

It is interesting to note that the variance of the restricted estimate is even larger than the unconditional variance of y_0. This is possible as $(w_1, w_2) = (0, 0)$ is not included in the restricted parameter space.

So far we argued and showed illustratively, that an unrestricted OLS estimate can be superior to the constraint SC estimate in settings with few panel units and a clear correlation structure among the units. This indication will be further refined in subsequent Monte Carlo simulations. In microeconometric settings it is usually assumed that the units

[7] The derivation of the employed estimators is postponed to the appendix.

in the treatment group and units in the control group are uncorrelated. In such cases the construction of a SC is unpromising as the dependency between treatment unit and donors is the core condition for a plausible estimation of the counterfactual. In macroeconomic applications however, the variables in the treatment and control group (e.g. GDP) are typically correlated and it is therefore important to model this relationship. As the simple scenario with only two panel units in the donor pool is highly unrealistic in practice, we now move to the general static case with $J + 1$ panel units.

3.3. General Static Extension

In empirical macroeconomic practice, the quantities of interest are commonly measured at monthly or quarterly intervals, typically not at a coarser resolution. Hence, it is frequently observed that the number of pre-intervention time periods $(T_0 - 1)$ is relatively small and may even be smaller than the number of units in the donor pool J. In such scenarios, the unrestricted OLS estimate may face issues of instability or, in the case of $T_0 - 1 < J$, due to singularity, it may not even be identified. ADH solve this issue by restricting the weights to be non-negative and to sum up to one. This regularization is essential as it prevents the method from overfitting the pre-treatment data and guarantees the existence of unique weights, especially when dealing with a small number of pre-treatment periods. However, alternative methods are also capable of ensuring weight stability and generalizability. For example, [Doudchenko and Imbens, 2016] suggest using an elastic net regression to regularize the donor weights and obtain comparable estimated treatment effects for different empirical applications of SC. Their objective function has the following form:

$$\widehat{w} = \arg\min_{v} \sum_{t=1}^{T_0-1} \left(y_{0,t} - \mu^* - \sum_{j=1}^{J} w_j y_{j,t} \right)^2 + \lambda_1 \left(\sum_{j=1}^{J} w_j^2 \right) + \lambda_2 \left(\sum_{j=1}^{J} |w_j| \right)$$

there are also other competing procedures to ensure weight stability and generazibility like the elastic net for synthetic controls as proposed by .

Therefore some kind of regularization is necessary to obtain a reliable estimate of $\widehat{Y}_0^N$.

$$Q(w, \lambda_1, \lambda_2) = \sum_{t=1}^{T_0-1} \left(y_{0,t} - \mu^* - \sum_{j=1}^{J} w_j y_{j,t} \right)^2 + \lambda_1 \left(\sum_{j=1}^{J} w_j^2 \right) + \lambda_2 \left(1 - \sum_{j=1}^{J} w_j \right)$$

3.4. Univariate Dynamic Extension

What must be clear by now

- **TBD**

When modeling macroeconomic time series it is often assumed that the $(k+1) \times 1$ vector of time series $y_t = (Y_{0t}, ..., Y_{kt})'$ can be represented by a VAR model given by

3.5. Multivariate Dynamic Extension

4. Simulation

4.1. Static Data Generating Processes

Simulation-Procedure

- [Abadie et al., 2010] suppose that the counterfactuals $Y_{1,t}^N$ for $t > T_0$ are given by a factor model of the form $Y_{i,t}^N = \alpha_t + \theta_t Z_i + \lambda_t \mu_i + \epsilon_{it}$. α_t denotes an unknown common factor with constant loadings across panel units. Z_i is a vector of observed panel-specific covariates, θ_t is a vector of unknown parameters, λ_t is a vector of unknown common facots and μ_i are panel-specific unknown factor loadings. The unobserved transitory shocks ϵ_{it} have zero mean at the panel level. For this specific setting, [Abadie et al., 2010] show that "[...] the bias of the SC-estimator can be bounded by a function that goes to zero as the number of pre-treatment periods increases." Further the number of donor units has to be fixed.

- [Ferman, 2021] considers a de-meaned scenario without additional covariates. The representation of the counterfactuals therefore boils down to $Y_{i,t}^N = \lambda_t \mu_i + \epsilon_{it}$. Thus the counterfactual is given by the composition of the unknown panel-specific factor loadings and the unknown common factors plus the idiosyncratic shocks.

- We re-estimated the Tobacco Control and the Basque application and found that the inclusion of additional covariates did not improve the predictive accuracy of the SC estimator. Therefore, we follow the simulation suggestion of [Ferman, 2021] and root our simulation in his proposed factor model without additional covariates.

- Analogous to Ferman, we consider a setting with two common factors, $\lambda_{1,t}$ and $\lambda_{2,t}$. The potential outcomes for the treated unit and for the first half of the donor pool load exclusively with loading one on the first factor, the remaining donors load exclusively with loading one on the second factor. Therefore μ_i is a (2×1)-rowvector with the first (second) entry being one and the second (first) entry being zero for the first (second) half of the donor pool.

- In each simulation, we simulate a total of $T_0 + T_1$ observations, where T_0 represents the length of the pre- and T_1 the length of the post-treatment period. In order to have a simulation framework that is as close as possible to real world SC-applications, we choose $T_0 \in \{20, 50, 100\}$ and $T_1 \in \{10, 20, 30\}$. Further, we define the size of the donor pool as $J \in \{5, 10, 15, 20, 25, 30\}$.

- To initialize the simulation, we simulate the two common factors by drawing $T_0 + T_1$ independent and identically distributed (iid) observations from a standard normal distribution. Next, we multiply the common factors for all donors and the treatmend unit with the factor loadings μ_i and add iid standard normal shocks. Additionally, we add panel-specific iid standard normal intercepts such that our DGP takes the following form: $Y_{i,t}^N = \gamma_i + \lambda_t \mu_i + \epsilon_{it}$.
- Figure 2 visualizes one potential DGP with $T_0 = 20$ and $T_1 = 10$. To make the factor structure visible, we scaled the factor variance by 10^1 and the shock variance by 10^{-1}. Moreover, for better eyeball inspection, we added a constant treatment effect of $\delta_{1,t} = 10$ for $t > T_0$. Note that the specific nature of the treatment effect is irrelevant for our investigation. Since $Y_{1,t}^T$ is observable for $T > T_0$, we can choose an arbitrary functional form or even implement no treatment effect at all.

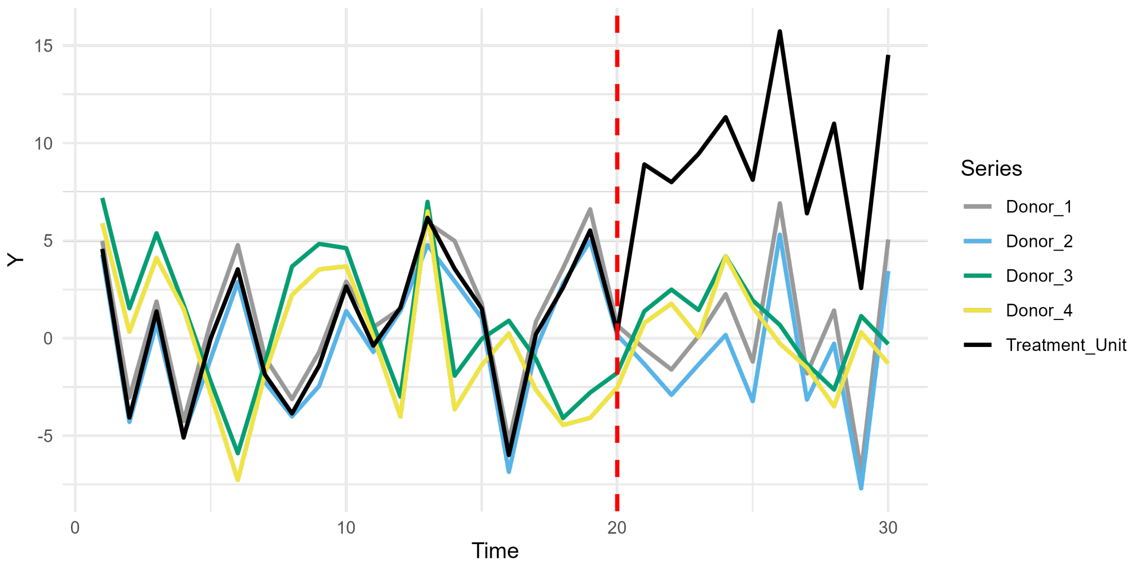

Figure 2. Example Factor-DGP

- The described factor structure is inherent in Figure 2: The treatment unit and the first half of the donors (Donor_1 and Donor_2) as well as the second half of the donors (Donor_3 and Donor_4) share a common factor structure. The constant treatment effect is added after $T_0 = 20$ periods, resulting in a 10 unit vertical shift of the treatment series.

- We consider five different models whose goal is to recover the true factor structure of the treatment unit in the pre-treatment period and to predict the counterfactual in the post-treatment period as accurately as possible. Put differently, we train the models on T_0 pre-treatment observations and test their performance using T_1 post-treatment observations.

- The following models are employed: **(Update when previous parts are written)**

  1. Factor-Model (FACTOR): It is assumed that the common factors and the idiosyncratic component are uncorrelated and the idiosyncratic errors are mutually uncorrelated. This suggests to estimate the common factors as linear function of the donors. A popular estimator with this property is the Principal Components (PC) estimator. In the training period, we obtain the predictions by regressing the treatment series on the "latent" factors. As we implemented a two-factor structure, the factors are computed by multiplying the first two eigenvalues of the covariates covariance matrix with the matrix of the covariates. The forecasts for the testing period are obtained by multiplying the factor structure of the post-treatment period with the regression coefficients of the pre-treatment regression. This model is most natural when generating data according to a factor process. Therefore, we expect it to perform best among all models.
  2. Synthetic-Control-Model (SC): In case of no additional explanatory variables, the SC-approach reduces to a constraint regression that regresses the treatment series on the donor series given the constraint of no intercept and non-negative coefficients that sum up to one. ADH argue that the constraints prevent the SC models from over-fitting, but they do so at the cost of a reduction in flexibility.
  3. Regularized OLS-Model (REGOLS): Our proposed model. We allow for a constant and arbitrary coefficient values. We propose a two-dimensional regularization: A ridge/l2-norm penalty that penalizes the coefficient sum and a

second penalty term that shrinks the coefficient sum towards one. We identify the optimal hyperparameter-combination by applying a 50/50 train-test-split in the pre-treatment period on a two-step random grid search. In the first step, we randomly select 400 hyperparameter-combinations from a (50×50)-grid. In the second step, we enclose the potential optimum by sequentially holding the first and the second hyperparameter fixed while increasing and decreasing the remaining hyperparameter on a coarser grid.

4. Elastic Net (NET): [Doudchenko and Imbens, 2016] propose to estimate the counterfactual using an elastic net regression.

5. OLS-Model (OLS): Finally, to verify the belief that a simple linear model overfits the training data and therefore yields poor forecasting accuracy in the testing period, we also employ an ordinary least squares model.

All simulations are conducted with 1,000 iterations per combination of pre- and post-treatment horizon and donor quantity.

Table S1. Simulation Results of the Static Factor Model with $\mathbf{J} = \mathbf{5}$ Donors.

T_0	T_1	FACTOR	SC	REGOLS	NET	OLS
		RMSE (BIAS) [VARIANCE]	RMSE (BIAS) [VARIANCE]	RMSE (BIAS) [VARIANCE]	RMSE (BIAS) [VARIANCE]	RMSE (BIAS) [VARIANCE]
20	30	1.2494	1.4604	1.2942	1.2959	1.3552
		(0.0166)	(0.0001)	(0.0142)	(0.0123)	(0.0102)
		[0.6158]	[1.1078]	[0.6464]	[0.5695]	[1.1093]
	20	1.2595	1.4560	1.3080	1.3067	1.3665
		(-0.0068)	(0.0320)	(-0.0009)	(-0.0003)	(0.0066)
		[0.6477]	[1.0652]	[0.6610]	[0.5948]	[1.0979]
	10	1.2342	1.4322	1.2877	1.2865	1.3569
		(0.0250)	(0.0256)	(0.0256)	(0.0202)	(0.0309)
		[0.6312]	[1.0473]	[0.6361]	[0.5516]	[1.0745]
50	30	1.1819	1.4327	1.2130	1.1987	1.2133
		(-0.0025)	(-0.0015)	(-0.0036)	(-0.0014)	(-0.0034)
		[0.6326]	[1.0579]	[0.6444]	[0.5430]	[0.7755]
	20	1.1837	1.4224	1.2124	1.2046	1.2194
		(0.0092)	(0.0097)	(0.0144)	(0.0131)	(0.0150)
		[0.6469]	[1.0563]	[0.6396]	[0.5583]	[0.7840]
	10	1.1663	1.3990	1.1966	1.1815	1.1968
		(0.0038)	(-0.0019)	(-0.0041)	(0.0045)	(-0.0014)
		[0.5825]	[0.9754]	[0.5731]	[0.5071]	[0.7163]
100	30	1.1636	1.3944	1.1818	1.1746	1.1807
		(-0.0046)	(0.0176)	(-0.0060)	(-0.0052)	(-0.0057)
		[0.6505]	[1.0736]	[0.6336]	[0.5802]	[0.7192]
	20	1.1684	1.3973	1.1861	1.1808	1.1862
		(-0.0009)	(0.0049)	(-0.0024)	(-0.0020)	(-0.0022)
		[0.6201]	[1.0416]	[0.5941]	[0.5488]	[0.6838]
	10	1.1434	1.3686	1.1552	1.1528	1.1557
		(0.0127)	(0.0263)	(0.0114)	(0.0123)	(0.0129)
		[0.5850]	[0.9784]	[0.5811]	[0.5151]	[0.6471]

Table S2. Simulation Results of the Static Factor Model with $\mathbf{J = 10}$ Donors.

T_0	T_1	**FACTOR**	**SC**	**REGOLS**	**NET**	**OLS**
		RMSE (BIAS) [VARIANCE]	RMSE (BIAS) [VARIANCE]	RMSE (BIAS) [VARIANCE]	RMSE (BIAS) [VARIANCE]	RMSE (BIAS) [VARIANCE]
20	30	1.1613 (-0.0051) [0.7638]	1.2867 (-0.0034) [1.0520]	1.2304 (-0.0040) [0.7474]	1.2739 (-0.0057) [0.7799]	1.6198 (-0.0090) [2.1449]
	20	1.1555 (-0.0059) [0.7316]	1.2840 (-0.0267) [1.0055]	1.2250 (-0.0059) [0.7538]	1.2595 (0.0061) [0.7655]	1.6167 (-0.0024) [2.1752]
	10	1.1312 (-0.0091) [0.6970]	1.2532 (-0.0102) [0.9745]	1.1987 (-0.0140) [0.7263]	1.2395 (-0.0149) [0.7504]	1.5811 (-0.0052) [2.0458]
50	30	1.1043 (0.0071) [0.8100]	1.2497 (0.0032) [1.0271]	1.1369 (0.0109) [0.7619]	1.1467 (0.0111) [0.6834]	1.2086 (0.0083) [1.1120]
	20	1.0966 (0.0079) [0.7854]	1.2308 (-0.0060) [1.0024]	1.1317 (0.0024) [0.7350]	1.1407 (0.0063) [0.6546]	1.2074 (0.0048) [1.0766]
	10	1.0912 (-0.0076) [0.7318]	1.2167 (-0.0053) [0.9551]	1.1204 (-0.0075) [0.7016]	1.1281 (-0.0058) [0.6186]	1.1895 (0.0001) [1.0395]
100	30	1.0851 (0.0065) [0.7875]	1.2316 (0.0110) [1.0178]	1.1063 (0.0067) [0.7389]	1.1112 (0.0080) [0.6770]	1.1323 (0.0081) [0.9248]
	20	1.0733 (-0.0137) [0.7698]	1.2088 (0.0019) [0.9747]	1.0917 (-0.0134) [0.7290]	1.0941 (-0.0125) [0.6631]	1.1202 (-0.0122) [0.9043]
	10	1.0661 (-0.0082) [0.7524]	1.1986 (-0.0119) [0.9553]	1.0895 (-0.0096) [0.7098]	1.0934 (-0.0129) [0.6451]	1.1178 (-0.0113) [0.8864]

Table S3. Simulation Results of the Static Factor Model with $\mathbf{J} = \mathbf{15}$ Donors.

T_0	T_1	FACTOR	SC	REGOLS	NET	OLS
		RMSE (BIAS) [VARIANCE]	RMSE (BIAS) [VARIANCE]	RMSE (BIAS) [VARIANCE]	RMSE (BIAS) [VARIANCE]	RMSE (BIAS) [VARIANCE]
20	30	1.1355 (0.0015) [0.7829]	1.2504 (-0.0022) [1.0159]	1.2171 (0.0004) [0.7942]	1.3089 (0.0012) [1.0400]	2.4509 (0.0009) [6.0410]
	20	1.1223 (0.0082) [0.7681]	1.2368 (-0.0057) [1.0051]	1.2088 (0.0130) [0.8111]	1.3010 (0.0024) [1.1466]	2.4339 (0.0089) [6.0128]
	10	1.1199 (0.0060) [0.7029]	1.2299 (0.0152) [0.9262]	1.2142 (-0.0096) [0.7565]	1.3095 (-0.0061) [1.0792]	2.4066 (-0.0104) [5.7301]
50	30	1.0834 (0.0062) [0.8168]	1.2104 (0.0041) [0.9866]	1.1219 (0.0041) [0.7623]	1.1337 (0.0041) [0.6970]	1.2756 (0.0005) [1.3377]
	20	1.0794 (0.0047) [0.7768]	1.2016 (0.0050) [0.9709]	1.1222 (0.0043) [0.7428]	1.1324 (0.0054) [0.6599]	1.2737 (0.0025) [1.2865]
	10	1.0674 (0.0001) [0.7333]	1.1873 (-0.0077) [0.8756]	1.1010 (0.0021) [0.6879]	1.1105 (0.0026) [0.6355]	1.2532 (0.0028) [1.2290]
100	30	1.0732 (0.0036) [0.8262]	1.1780 (0.0093) [0.9729]	1.0966 (0.0043) [0.7689]	1.1032 (0.0062) [0.6935]	1.1508 (0.0059) [1.0253]
	20	1.0645 (-0.0153) [0.8304]	1.1774 (-0.0091) [0.9807]	1.0905 (-0.0185) [0.7736]	1.0963 (-0.0192) [0.7014]	1.1459 (-0.0203) [1.0300]
	10	1.0627 (0.0114) [0.7628]	1.1613 (0.0090) [0.9068]	1.0829 (0.0146) [0.7123]	1.0902 (0.0149) [0.6393]	1.1378 (0.0171) [0.9584]

Table S4. Simulation Results of the Static Factor Model with $\mathbf{J} = \mathbf{20}$ Donors.

T_0	T_1	FACTOR	SC	REGOLS	NET	OLS
		RMSE (BIAS) [VARIANCE]	RMSE (BIAS) [VARIANCE]	RMSE (BIAS) [VARIANCE]	RMSE (BIAS) [VARIANCE]	RMSE (BIAS) [VARIANCE]
20	30	1.1278	1.2332	1.2183	1.2859	NA
		(0.0020)	(-0.0040)	(-0.0039)	(-0.0079)	NA
		[0.7964]	[1.0193]	[0.8413]	[0.9901]	NA
	20	1.1244	1.2312	1.2083	1.2712	NA
		(-0.0082)	(0.0043)	(-0.0054)	(0.0025)	NA
		[0.7614]	[0.9685]	[0.7839]	[0.9067]	NA
	10	1.1054	1.1986	1.1987	1.2632	NA
		(0.0172)	(0.0063)	(0.0171)	(0.0169)	NA
		[0.7525]	[0.9464]	[0.8319]	[0.9309]	NA
50	30	1.0703	1.1749	1.1087	1.1247	1.3707
		(-0.0056)	(-0.0160)	(-0.0070)	(-0.0088)	(-0.0067)
		[0.8408]	[0.9813]	[0.7851]	[0.7387]	[1.6546]
	20	1.0678	1.1720	1.1117	1.1237	1.3646
		(0.0207)	(0.0202)	(0.0149)	(0.0194)	(0.0054)
		[0.8154]	[0.9649]	[0.7801]	[0.7150]	[1.5848]
	10	1.0514	1.1444	1.0846	1.0976	1.3227
		(-0.0141)	(-0.0264)	(-0.0120)	(-0.0085)	(-0.0089)
		[0.7774]	[0.8997]	[0.7324]	[0.6672]	[1.4695]
100	30	1.0447	1.1422	1.0679	1.0793	1.1626
		(-0.0039)	(0.0168)	(-0.0029)	(-0.0007)	(-0.0016)
		[0.8600]	[0.9745]	[0.7904]	[0.7231]	[1.1338]
	20	1.0510	1.1476	1.0715	1.0815	1.1589
		(-0.0144)	(-0.0185)	(-0.0139)	(-0.0105)	(-0.0113)
		[0.8576]	[0.9550]	[0.7864]	[0.7234]	[1.1391]
	10	1.0304	1.1144	1.0505	1.0598	1.1397
		(0.0223)	(0.0166)	(0.0271)	(0.0270)	(0.0321)
		[0.8118]	[0.9018]	[0.7425]	[0.6826]	[1.0761]

Table S5. Simulation Results of the Static Factor Model with $\mathbf{J = 25}$ Donors.

T_0	T_1	FACTOR	SC	REGOLS	NET	OLS
		RMSE (BIAS) [VARIANCE]	RMSE (BIAS) [VARIANCE]	RMSE (BIAS) [VARIANCE]	RMSE (BIAS) [VARIANCE]	RMSE (BIAS) [VARIANCE]
20	30	1.1093 (0.0013) [0.7989]	NA NA NA	1.2010 (0.0090) [0.8648]	1.2554 (0.0116) [0.9421]	NA NA NA
	20	1.1073 (0.0131) [0.8177]	NA NA NA	1.1989 (0.0108) [0.8516]	1.2708 (0.0093) [0.9724]	NA NA NA
	10	1.0971 (-0.0046) [0.7589]	NA NA NA	1.1928 (-0.0001) [0.8153]	1.2447 (0.0013) [0.8945]	NA NA NA
50	30	1.0597 (-0.0018) [0.8466]	1.1582 (0.0135) [0.9590]	1.0996 (-0.0048) [0.7879]	1.1172 (-0.0079) [0.7353]	1.4863 (-0.0151) [1.9825]
	20	1.0538 (-0.0150) [0.8350]	1.1525 (0.0043) [0.9332]	1.0993 (-0.0082) [0.7821]	1.1139 (-0.0101) [0.7263]	1.4811 (0.0033) [1.9856]
	10	1.0432 (0.0062) [0.8077]	1.1428 (-0.0038) [0.9111]	1.0831 (0.0062) [0.7625]	1.0997 (0.0064) [0.7214]	1.4400 (-0.0066) [1.8603]
100	30	1.0378 (0.0000) [0.8501]	1.1232 (0.0058) [0.9454]	1.0641 (-0.0003) [0.7912]	1.0741 (0.0006) [0.7149]	1.1959 (0.0044) [1.2244]
	20	1.0483 (0.0110) [0.8655]	1.1401 (0.0130) [0.9457]	1.0747 (0.0114) [0.7968]	1.0842 (0.0127) [0.7234]	1.2002 (0.0147) [1.2330]
	10	1.0296 (-0.0018) [0.8083]	1.1154 (-0.0017) [0.8879]	1.0551 (-0.0012) [0.7444]	1.0648 (0.0001) [0.6782]	1.1811 (-0.0040) [1.1463]

Table S6. Simulation Results of the Static Factor Model with $\mathbf{J} = \mathbf{30}$ Donors.

T_0	T_1	FACTOR	SC	REGOLS	NET	OLS
		RMSE	RMSE	RMSE	RMSE	RMSE
		(BIAS)	(BIAS)	(BIAS)	(BIAS)	(BIAS)
		[VARIANCE]	[VARIANCE]	[VARIANCE]	[VARIANCE]	[VARIANCE]
20	30	1.1013	NA	1.1899	1.2523	NA
		(0.0128)	NA	(0.0149)	(0.0115)	NA
		[0.8224]	NA	[0.8865]	[0.9715]	NA
	20	1.1043	NA	1.1825	1.2442	NA
		(0.0014)	NA	(0.0031)	(0.0052)	NA
		[0.8072]	NA	[0.8431]	[0.9420]	NA
	10	1.0890	NA	1.1647	1.2337	NA
		(-0.0075)	NA	(-0.0054)	(-0.0055)	NA
		[0.7761]	NA	[0.8107]	[0.8943]	NA
50	30	1.0622	1.1477	1.1039	1.1297	1.6851
		(-0.0022)	(0.0062)	(0.0009)	(-0.0012)	(0.0059)
		[0.8658]	[0.9630]	[0.8137]	[0.7852]	[2.6638]
	20	1.0508	1.1372	1.0936	1.1190	1.6716
		(-0.0016)	(-0.0084)	(-0.0009)	(-0.0004)	(-0.0006)
		[0.8700]	[0.9523]	[0.8184]	[0.7841]	[2.5964]
	10	1.0445	1.1342	1.0880	1.1061	1.6681
		(0.0006)	(-0.0043)	(0.0052)	(0.0091)	(0.0174)
		[0.8279]	[0.9218]	[0.7784]	[0.7648]	[2.5590]
100	30	1.0420	1.1178	1.0662	1.0793	1.2394
		(0.0097)	(0.0172)	(0.0107)	(0.0094)	(0.0070)
		[0.8864]	[0.9693]	[0.8258]	[0.7565]	[1.3694]
	20	1.0435	1.1237	1.0669	1.0788	1.2351
		(0.0072)	(0.0038)	(0.0054)	(0.0057)	(0.0064)
		[0.8778]	[0.9520]	[0.8077]	[0.7417]	[1.3400]
	10	1.0159	1.0890	1.0410	1.0465	1.2026
		(0.0027)	(0.0173)	(0.0041)	(0.0020)	(0.0038)
		[0.8381]	[0.8876]	[0.7678]	[0.7076]	[1.2823]

4.2. Weakly Dynamic Data Generating Processes

4.3. Dynamic Data Generating Processes

5. Applications

We consider three leading examples:

- [Abadie and Gardeazabal, 2003]
- [Abadie et al., 2010]
- [Abadie et al., 2015]

6. Conclusion

- Some concluding remark and an outlook
- Keep short, around 1-2 pages
- Natural extension: case with explanatory variables

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Appendix

Simple Static Extension

OLS Solution

In case the population covariance matrix is observable, the OLS-coefficients can be directly derived from it: $\left(w_1^{OLS}, w_2^{OLS}\right) = \boldsymbol{\Sigma_2}^{-1}\boldsymbol{\sigma_{12}}$

SC Solution

The restricted solution is can directly be derived from the covariance matrix. The first index in the square brackets indicates the row, the second the column position.

$$\begin{aligned} w_1^{SC} &= (\boldsymbol{\sigma_{12}}'[1] - \boldsymbol{\sigma_{12}}'[2] - \boldsymbol{\Sigma_2}[2,1] + \boldsymbol{\Sigma_2}[1,1]) \,/\, (\boldsymbol{\Sigma_2}[1,1] + \boldsymbol{\Sigma_2}[2,2] - 2 * \boldsymbol{\Sigma_2}[1,2]) \\ &= (0.1 - 0.4 - 0.5 + 1)/(1 + 1 - 2 * 0.5) = 0.2 \end{aligned}$$

$$\begin{aligned} w_2^{SC} &= (\boldsymbol{\sigma_{12}}'[2] - \boldsymbol{\sigma_{12}}'[1] - \boldsymbol{\Sigma_2}[1,2] + \boldsymbol{\Sigma_2}[2,2]) \,/\, (\boldsymbol{\Sigma_2}[2,2] + \boldsymbol{\Sigma_2}[1,1] - 2 * \boldsymbol{\Sigma_2}[2,1]) \\ &= (0.4 - 0.1 - 0.5 + 1)/(1 + 1 - 2 * 0.5) = 0.8 \end{aligned}$$

Variances

The variances are derived from the weights and the covariance matrix:

$$\begin{aligned} var(Y_0 - w_1 Y_1 - w_2 Y_2) = {} & var(Y_0) + w_1^2 \cdot var(Y_1) + w_2^2 \cdot var(Y_2) \, - \\ & 2 \cdot w_1 \cdot cov(Y_0, Y_1) - 2 \cdot w_2 \cdot cov(Y_0, Y_2) \, + \\ & 2 \cdot w_1 w_2 \cdot cov(Y_1, Y_2) \end{aligned}$$